

Modern Concepts of Cardiovascular Disease

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PULMONARY COMPLICATIONS IN HEART DISEASE

Because of inherent difficulties in diagnosis or through oversight, too many pulmonary conditions associated with heart disease first come to light at the autopsy table. In an effort to reduce our own errors in diagnosis, we consider the following conditions which we believe offer the greatest challenge: 1) Pulmonary Embolism, 2) Pleural Effusion, 3) Pulmonary Atelectasis, 4) Pulmonary Congestion, Hypostatic Congestion, 5) Hypostatic Pneumonia, 6) Pulmonary Edema; Acute, Chronic, and 7) Hemoptysis.

Pulmonary Embolism

One of the most serious complications of heart disease is pulmonary embolism. Although the source of the embolus or emboli is usually the lower extremities, it is now certain that the heart itself is more frequently the site of origin than was formerly supposed. Intracardiac thrombi most often form in the left side of the heart and emboli when released go out through the systemic circulation with subsequent blocking of small arteries in the extremities, viscera, or brain. However, mural thrombi in the right heart are by no means rare. They occur when coronary thrombosis produces septal or right ventricular infarcts. Bacterial endocarditis or verrucous endocarditis as seen in Libman-Sacks Disease may give rise to multiple emboli which lodge in the lungs. In auricular fibrillation especially if associated with mitral stenosis, eddying currents of blood may form clots in the dilated right auricular appendage which find their way to the lungs. The shock state with dilatation of the heart and protracted low blood pressure seen in the patient with a large coronary vessel occlusion is conducive to the formation of

thrombi. These may break loose and become emboli in the pulmonary circulation.

Pulmonary emboli which come from the veins in the legs in patients with seriously damaged hearts are due to slowing of the circulation. Methods to discourage their formation should be instituted almost as soon as these individuals are put to bed. Passive exercises such as flexing the ankles, knees, and thighs several times each day may be all that is necessary to prevent the development of a serious or fatal lung complication.

In all cardiac conditions in which emboli might be anticipated, the use of the anticoagulants is strongly recommended but only if proper laboratory controls are available. At the present time dicumarol (sometimes following heparin therapy) is the drug of choice. Apart from diseases of the liver which might alter the prothrombin time, there are few contraindications for anticoagulant therapy. If dicumarol is used, a laboratory qualified to perform frequent prothrombin tests on the blood is essential. Deaths from hemorrhage have occurred when the anticoagulants are used without careful laboratory checks.

The differential diagnosis between pulmonary emboli arising from a location in the peripheral circulation and acute coronary thrombosis is not an easy one to make especially in the first few hours. Serial electrocardiograms may furnish the only method for making this differentiation. A thorough search for venous thrombosis in the extremities by inspection, palpation, and the use of the Homan's sign may give the answer.

There is another aspect to the problem of

ANNUAL MEETING

The Annual Meeting and Twenty-Fourth Scientific Session of the American Heart Association will be held at the Chalfonte-Haddon Hall Hotel, Atlantic City, New Jersey, June 7-10, 1951. All those desiring to attend should make room reservations directly with hotels in Atlantic City at the earliest possible date.

correct differential diagnosis. It is the fact that the myocardial anoxia associated with large pulmonary emboli may produce a coronary type of electrocardiogram in individuals who already have advanced coronary insufficiency. The recognition of this possibility may save embarrassment and justify a delay in making an early positive diagnosis.

Pleural Effusion

Fluid in the pleural cavity is a complication of congestive heart failure in so many patients that this condition should be suspected if cough and dyspnea persist in spite of thorough treatment with the usual remedies. Unfortunately, some of the physical signs are not easily elicited. Shifting dullness and absent breath sounds, the classical signs, may not be present. This is particularly true if there is a large effusion under which circumstances tubular breathing may suggest parenchymal lung disease. Old pleural fibrosis at either lung base may obscure a sizable effusion or lead to a misdiagnosis of fluid. Even x-ray studies made in the usual manner may be misleading. A posterior-anterior view of the chest often fails to reveal a significant amount of fluid back of the dome of the diaphragm which might have been discovered if lateral views had been made. Another physical difficulty in x-ray interpretation arises when the fluid is held in a thin film extending evenly up along the chest wall in some cases as high as the apex of the lung. This gives a hazy appearance but no fluid level is seen. Lateral as well as posterior-anterior views and thorough fluoroscopic studies are indicated when clinical judgment points to unverified pleural effusion. Occasionally, a diagnostic thoracentesis will reveal the presence of fluid when all other methods fail. The dramatic relief of dyspnea when relatively small amounts of fluid are removed from the chest justifies a thorough search for effusion. Prompt thoracentesis may mark the beginning of real improvement in this distressing condition. It is not advisable to withdraw the entire amount lest a painful pleurisy develop. The patient with congestive failure whose symptoms suggest that complete rest is necessary should not be kept in bed during the first few days, for absolute confinement favors the rapid development of pleural effusion.

Pulmonary Atelectasis

Pulmonary atelectasis is one of the most commonly unrecognized complications of heart disease. Atelectatic areas vary in size from microscopic points to larger areas

which may coalesce finally involving a major portion of a lobe.

Important causative factors are: 1) Anoxia in the very ill cardiac especially in the debilitated individual, 2) Failure to clear the finer air passages of fluid and detritus by reason of inability to cough long enough or hard enough to evacuate this material, 3) Long pauses in the respiratory cycle as in Cheyne-Stokes breathing, 4) Very weak respiratory excursions so frequently seen in the acutely shocked patient with myocardial infarction, and 5) The use of opiates with the subsequent depression of respiratory movements.

It is of the utmost importance that atelectatic areas be recognized promptly before they increase in size, become fixed, or infected. Early diagnosis is not always possible but if any of the situations described exist, preventive steps should be practiced. These consist of: Frequent changes in the position of the patient, gently rolling him from side to side; having him sit up in bed several times each day; unless too ill, having him dangle his feet over the side of the bed; encouraging deep breathing, aiding him if necessary with mild artificial respiration maneuvers; urging him to cough several times every three hours; and, administering 100 per cent oxygen by mask after coughing. When the first sure signs of focal atelectasis appear, more vigorous measures are indicated.

It is almost certain that by the time areas of atelectasis are large enough to become fixed in one spot, or do not clear when the patient coughs, or when roentgen examination shows airless areas or elevation of the diaphragm, the process has progressed to the stage where pneumonitis is present.

The physical signs produced by focal pulmonary atelectasis are extremely variable and may be impossible to diagnose clinically or by x-ray. The condition, however, can be suspected and in a measure prevented before small areas of atelectasis coalesce to form larger areas which may in turn become infected and result in a real pneumonia. Penicillin given at this stage may prevent a spread of the infection. Caffeine sodium benzoate (0.5 gram) every 4-6 hours for three or four doses may be given to increase the respiratory excursions. These measures plus oxygen inhalations given more constantly will favor aeration of the collapsed areas. We are attracted by the possibility of applying to the cardiacs the idea outlined by Swank¹ who used the Drinker Respirator to prevent atelectasis in paralyzed patients.

The small patchy areas of pneumonia reported in the progress notes of patients with

¹ Swank, R. L. *Ann. Int. Med.* 32:229:1950

congestive heart failure are frequently areas of atelectasis which disappear if the patient is moved, if he coughs, or if oxygen is administered. Such areas may form in some part of the lung where they may not be recognized and there coalesce, become infected, and produce the so called "terminal pneumonia" which is so resistant to penicillin and sulfa treatment.

Pulmonary Congestion

When there is obstruction to the flow of blood in the pulmonary circulation, passive congestion can be said to be present. That it occurs in valvular and myocardial insufficiency from whatever cause is well known. In the early stages the lungs are simply engorged. If this persists the lungs actually undergo structural changes by reason of connective tissue formation and are less efficient organs for the transfer of oxygen.

Passive congestion of the lungs is often not recognized or even suspected until *intra-alveolar* edema appears. The fact that there may be *inter-alveolar* edema of a degree sufficient to produce distressing symptoms must be kept in mind. The cardinal clinical findings are dyspnea, cough, and cyanosis but even before these are evident, the presence of pulmonary congestion should be suspected if the vital capacity decreases. If the patient resting in bed has an appreciable diminution of his vital capacity, it can be predicted that he will develop dyspnea. Now that a portable vital capacity apparatus is simply designed and is accurate enough for practical purposes, one can get a great deal of information concerning the degree of pulmonary congestion and the severity of dyspnea by frequent estimations of the vital capacity. Due to the fact that the lungs are in a relatively non-elastic cage, it is evident that they cannot contain as much air when they become engorged with blood. When this occurs, symptoms appear. On physical examination, the respiratory excursion is found to be limited, the breath sounds are often diminished particularly at the bases, and fine moist rales are audible. Although the character of the sputum is likely to suggest a pure bronchitis, if examined carefully it will be found to contain more albumin than the average sputum of true bronchitis. This is due to the fact that there is mixed with it considerable serous exudate from the alveoli. The presence of heart failure cells in the sputum is also a differential point particularly if the congestion has been of long standing and so-called brown induration of the lung is present. X-ray studies are of considerable help in confirming the diagnosis of pulmonary congestion. Unfortunately, in the early stages when the condition should be recognized, interpretation is difficult.

It is important to discover passive congestion early, establish a schedule of limited activity, and give adequate digitalis even if the heart's rhythm is normal. The addition of mercurial diuretics at regular intervals may prevent a truly disabling bout of heart failure.

Hypostatic Congestion

It is scarcely necessary to mention the fact that hypostatic congestion is likely to occur in debilitated patients who have circulatory difficulties and who are permitted to remain in one position for hours at a time. The dependent parts of the lungs become congested, the alveoli fill with serum, and the airless areas develop. Later, the lungs may become swollen, firm, and tough with a change in color to dark red (splenization). If the condition of the heart muscle can be improved, the congestion clears, and stasis disappears. If it persists too long, however, the process is irreversible.

Hypostatic Pneumonia

This occurs commonly in passive congestion if infection supervenes. The appearance of fever, leukocytosis, increase in cough, cyanosis, and dyspnea may be the signs which confirm the diagnosis. This condition is not as rapidly fatal as formerly because of the effectiveness of penicillin in combating the infection. The earlier the situation is diagnosed and appropriate remedies applied, the greater is the chance for improvement. It is certainly true that these patients, particularly the elderly, do better if they change position frequently. The chance of developing a serious type of pulmonary congestion is less if patients are permitted to sit at the bedside at least part of the day.

Pulmonary Edema

Edema of the lung results when the capillaries exude serous fluid into the alveoli or into the connective tissue (*interstitial edema*). In fact, fluid must first pass the barrier of the connective tissue before it reaches the alveoli. Edema of the lungs may result from long standing passive congestion or occur with appalling speed and without previous warning. There are no positive physical signs by which interstitial edema, as contrasted with *intra-alveolar* edema, can be diagnosed. Its presence can be suspected if there is an increasing loss of cardiac reserve and lowered vital capacity with dyspnea even though no rales are heard. Chronic or slowly forming pulmonary edema is often marked by the presence of blood in the sputum which is likely to be present in varying amounts. It is due to blood cells coming into the alveoli with the exuded fluid. This material by blocking the smaller air passageways may be the

cause of the so-called congestive infarcts. Irregular, relatively airless areas may be found in both lungs by physical and roentgen examination. These changes are most prominent in the bases. Areas of focal parenchymal edema, however, may be found in other parts of the lung. Edema has a tendency to occur around chronic lung changes such as the scars of interlobar pleurisy or old tuberculous lesions. Areas of edema change location suddenly, appearing and disappearing in an unpredictable and capricious manner.

Acute Pulmonary Edema

Although most commonly occurring in hypertension, acute pulmonary edema is seen also with advanced valvular heart disease. The onset is often abrupt, and of alarming severity. The dyspnea may be sudden and terrifying. The patient may be overcome by a flooding of exudate into the bronchi and die immediately. The sudden onset of agonizing dyspnea with a rush of frothy pink sputum so copious that the patient cannot cough fast enough to keep ahead of the onrush of fluid, presents one of the most important medical emergencies. The treatment indicated is oxygen under pressure, artificial respiration, blood letting, morphia, and vasodilators if hypertension exists. Conheim states, "It is well to remember that in the terminal stage of heart disease, the patient does not die of pulmonary edema but he has pulmonary edema because he is dying."

Hemoptysis

The pulmonary hypertension accompanying mitral stenosis of severe degree some-

times causes hemoptysis occasionally of a degree severe enough to produce a significant anemia. Occasionally, the bleeding may be severe enough in one sharp episode or so frequently repeated that transfusions of whole blood are indicated. When this occurs it is a true example of pulmonary apoplexy. This condition is frequently confused with the hemoptysis accompanying pulmonary embolism from peripheral sources or the emboli which so commonly accompany advanced mitral stenosis particularly if the auricles are fibrillating.

Comment

In exploring the subject under discussion, it has been difficult to determine just what conditions to include; for example, should cor pulmonale, acute or chronic or both, be considered as a pulmonary complication of heart disease for we miss this diagnosis frequently. Does emphysema rate a spot on the agenda? Should massive hiatus hernia with pulmonary pressure and cardiac embarrassment be discussed? Does rupture of an aortic aneurysm into a pulmonary vessel have a place? What about including pulmonary arteritis, endarteritis, or atherosclerosis? Is pleuritis accompanying pericarditis to be omitted? Should not rheumatic pneumonitis receive a full discussion?

It is apparent that there are many situations in which the line between primary pulmonary disease or primary cardiac disease is not clearly drawn.

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SCIENTIFIC PROGRAM JUNE, 1951

All those desiring to present papers at the Twenty-Fourth Scientific Sessions of the American Heart Association at the Chalfonte-Haddon Hall Hotel, Atlantic City, New Jersey, June 7-10, 1951, should submit abstracts (in triplicate) of the proposed presentation of not more than 300 words to the Medical Director, American Heart Association. The deadline for the receipt of abstracts is February 10, 1951. The Program Committee will be published in a forthcoming issue.

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